1: Diabetologia 1996 Dec;39(12):1662-7

Diabetologia

The DIAB-HYCAR Study.

Passa P, Chatellier G.

Diabetes Department, Saint Louis Hospital, Paris, France.

Microalbuminuria and proteinuria are strong independent predictors for increased cardiovascular mortality in non-insulin-dependent diabetic (NIDDM) patients. In such patients, angiotensin converting enzyme (ACE) inhibition improves the evolution of diabetic nephropathy; however, no data are currently available on the effects of such intervention on cardiovascular morbidity and mortality. The aim of the Diab-Hycar study is to test the hypothesis that ACE inhibition with a low daily dose of 1.25 mg ramipril, which has no significant effect on blood pressure, may reduce cardiovascular morbidity and/or mortality in normotensive or hypertensive NIDDM patients with persistent albuminuria. Selected and followed by general practitioners, 4000 patients will receive their usual oral antidiabetic treatment and if necessary antihypertensive treatment (ACE inhibitors excluded). In addition in a randomized, double-blind trial they will be given either a placebo or 1.25 mg ramipril daily. The follow-up is currently scheduled to last 3 years. The efficacy of ACE-inhibition will be assessed by the following major end-points: cardiovascular death, sudden death, myocardial infarction, stroke, renal replacement therapy. The Diab-Hycar study started on 3 February 1995 By 1 September 1995, 11,000 urine samples were tested. The prevalence of persistent albuminuria was 23%, 964 patients were initially included in the study, with 619 eligible patients included soon after. Different strategies have been developed to record cardiovascular events correctly and to minimize the number of patients lost to follow-up.

Publication Types:
- Clinical Trial
- Randomized Controlled Trial

PMID: 9081852 [PubMed - indexed for MEDLINE]

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1: Cardiology 1997 Nov-Dec;88(6):548-55

Related Articles, Link

Early administration of ramipril in acute myocardial infarction: neurohormonal and hemodynamic effects and tolerability.

van der Ent M, Remme WJ, Bartels GL, Kruijssen DA, Krauss XH, van Hoogenhuyze DC.

Sticares Cardiovascular Research Foundation, Rotterdam, The Netherlands.

Although several large studies indicate a beneficial effect of angiotensin-converting enzyme (ACE) inhibitors after myocardial infarction, the optimal timing of therapy in terms of safety and the effects on neurohormones during myocardial infarction are less well known. In order to investigate the effect of ramipril, administered within 24 h after myocardial infarction, on hemodynamics and neurohormones and its safety, 20 patients with a myocardial infarction were studied. Nine patients had an anterior, 10 an inferior, and 1 a non-Q-wave infarction. Fourteen patients received thrombolytic therapy, whereas 6 did not. The initial dose of ramipril was 1.2: mg, but was gradually increased to 5 mg during the next 4 days. Side effects did not occur. The mean arterial pressure decreased 8 h after the first dose from 84 +/- 2 mm Hg (control) to 77 +/- 2 mm Hg ($p < 0.05$) and remained decreased thereafter. This was accompanied by a reduction in systemic resistance of 8% after 8 h and of 12% on day 2. Heart rate, cardiac and stroke indexes, and pulmonary artery and wedge pressures did not change. The ACE activity decreased within 1 h of ramipril administration with a maximum of 71% at 4 h after the second dose and remained at this level throughout the study. Angiotensin II decreased by 34% (day 2) and by 41% (day 5). The renin activity gradually increased from 33 +/- 7.5 to 75.4 +/- 11.5 microM/ml on day 5, whereas epinephrine was reduced from day 2 onwards, with a maximal reduction of 71% on day 5. Arginine vasopressin was significantly reduced 5 h after ramipril administration until the end of th study, with a maximum of 77% on day 3. Moreover, a late but significant decrease in norepinephrine occurred on day 5. Thus, oral ramipril results in early ACE inhibition, followed by progressive attenuation of the neuroendocrine activation and a reduction in afterload during the acute phase of myocardial infarction. It is well tolerated, also in combination with nitroglycerin and thrombolytic therapy.

PMID: 9397311 [PubMed - indexed for MEDLINE]

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1: Am J Kidney Dis 1996 Dec;28(6):832-40 Related Articles, Link

Effect of ramipril on blood pressure and protein excretion rate in normotensive nondiabetic patients with proteinuria.

Toto RD, Adams-Huet B, Fenves AZ, Mitchell HC, Mulcahy W, Smith RD.

Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, 75235-8856, USA.

Angiotensin-converting enzyme inhibitors reduce proteinuria in both normotensive and hypertensive patients with proteinuric renal disease. However, the mechanism of the antiproteinuric effect has not been clarified. We performed a prospective, double-blind, placebo-controlled, randomized crossover trial to test the hypothesis that the antiproteinuric effect of ramipril was due to an improvement in glomerular permselectivity independent of blood pressure and glomerular filtration rate. The effect of low-dose (1.25 mg/d) and high-dose (5 mg/d) ramipril was assessed in 15 normotensive nondiabetic patients with proteinuria (> 150 mg/d). The study was divided into four 12-week periods: placebo, high- or low-dose ramipril, crossover to low- or high-dose ramipril, and placebo. Blood pressure, glomerular filtratio rate, renal plasma flow rate, urinary protein excretion rate, and plasma angiotensin II levels were measured at the end of each period. Mean arterial pressure, urine protein to creatinine ratio, and albumin excretion rate decreased significantly during low- and high-dose ramipril. Glomerular filtration rate and renal plasma flow rate were not changed significantly. Plasma angiotensin II levels decreased with both low- and high-dose ramipril. There were no episodes of hypotension and only one subject developed cough during ramipril that did not require discontinuation of the study drug. In conclusion, administration of ramipril in both low and high doses lowered blood pressure and reduced proteinuria in this cohort of normotensive patients with a variety of proteinuric renal diseases. The antiproteinuric effect of ramipril is probably mediated by a reduction in glomerular capillary pressure.

Publication Types:
- Clinical Trial
- Randomized Controlled Trial

1: Circulation 1997 Nov 4;96(9):3164-72

Long-term ACE inhibition doubles lifespan of hypertensive rats

Linz W, Jessen T, Becker RH, Scholkens BA, Wiemer G.

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BACKGROUND: We compared the outcome of lifelong treatment with the ACE inhibitor ramipril in young prehypertensive stroke-prone spontaneously hypertensive rats (SHR-SP) and age-matched normotensive Wistar-Kyoto (WKY) rats. Ramipril was given in an antihypertensive and subantihypertensive dose. In addition to the primary end point, lifespan, surrogate parameters such as cardiac left ventricular hypertrophy, cardiac function and metabolism, and endothelial function were studied. METHODS AND RESULTS: One-month-old SHR-SP and WKY rats, 135 of each, were randomized into 3 groups. Each group was treated via drinking water with an antihypertensive high dose of ramipril (HRA, 1 mg x kg(-1) x d(-1)), a nonantihypertensive low dose of ramipril (LRA, 10 microg x kg(-1) x d(-1)), or placebo. Body weight and blood pressure were determined every 3 months. Molecular, biochemical, and functional data were assessed in SHR-SP and WKY rats after 15 and 30 months, respectively. These were the times when approximately 80% of the corresponding placebo group had died. Early-onset long-term ACE inhibition with HRA doubled lifespan to 30 months in SHR-SP, which was identical to the lifespan of placebo-treated normotensive WKY rats. LRA treatment prolonged lifespan from 15 to 18 months. In SHR-SP, left ventricular hypertrophy was completely prevented by HRA but not by LRA treatment. Cardiac function and metabolism as well as endothelial function were significantly improved by both doses of ramipril. Carotid expression of endothelial NO synthase was moderately enhanced, whereas cardiac ACE expression and activity were decreased to values of placebo-treated WKY rats. CONCLUSIONS: Lifelong ACE inhibition doubles lifespan in SHR-SP, matching that of normotensive WKY rats. This effect correlated with preservation of endothelial function, cardiac function/size, and metabolism. Thus, these data predict a beneficial outcome on survival in high-risk patients with hypertension and associated cardiovascular diseases by ACE inhibition.

1: Hypertension 1994 Apr;23(4):411-8

Related Articles, Link

Comment in:

- Hypertension. 1994 Apr;23(4):419-21.

Angiotensin-converting enzyme inhibition improves cardiac function. Role of bradykinin.

Gohlke P, Linz W, Scholkens BA, Kuwer I, Bartenbach S, Schnell A, Unger T.

Department of Pharmacology, Christian Albrechts University of Kiel, Germany.

The effect of chronic low- and high-dose treatment with the angiotensin-converting enzyme (ACE) inhibitor ramipril (0.01 and 1 mg/kg per day) on the development of hypertension and left ventricular hypertrophy as well as on functional and biochemical alterations of the heart was studied in stroke-prone spontaneously hypertensive rats treated prenatally and subsequently up to the age of 20 weeks. The contribution of endogenous bradykinin potentiation to the ACE inhibitor actions was assessed by cotreatment of rats with the bradykinin B2-receptor antagonist Hoe 140 (500 micrograms/kg per day SC) from 6 to 20 weeks of age. High- but not low-dose ACE inhibitor treatment prevented the development of hypertension and left ventricular hypertrophy. Chronic bradykinin receptor blockade did not attenuate the antihypertensive and antihypertrophic actions of ramipril. High-dose ramipril treatment improved cardiac function, as demonstrated by an increase in left ventricular pressure (29.9%), dP/dtmax (34.9%), and coronary flow (22.1%), without a change in heart rate. The activities of lactate dehydrogenase and creatine kinase and lactate concentration in the coronary effluent were reduced by 39.3%, 55.5%, and 66.7%, respectively. Myocardial tissue concentrations of glycogen and the energy-rich phosphates ATP and creatine phosphate were increased by 31.3%, 39.9%, and 73.7%, respectively, whereas lactate was decreased by 20.8%. Chronic low-dose ACE inhibitor treatment led to a pattern of changes in cardiodynamics and cardiac metabolism similar to that observed with the high dose. All ACE inhibitor-induced effects on cardiac function and metabolism were abolished by chronic bradykinin receptor blockade.(ABSTRACT TRUNCATED AT 250 WORDS)

1: Can J Cardiol 1996 Feb;12(2):127-37 Related Articles, Link

The HOPE (Heart Outcomes Prevention Evaluation) Study: the design of a large, simple randomized trial of an angiotensin-converting enzyme inhibitor (ramipril) and vitamin E in patients at high risk of cardiovascular events. The HOPE study investigators.

OBJECTIVE: To describe the design of the HOPE (Heart Outcomes Prevention Evaluation) study. DESIGN: Description of the key design features of HOPE, a large, simple randomized trial of two widely applicable treatments--ramipril, an angiotensin-converting enzyme inhibitor; and vitamin E, a naturally occurring antioxidant vitamin--in the prevention of myocardial infarction, stroke or cardiovascular death. SETTING: Two-hundred and sixty-seven hospitals, physician offices and clinics in Canada, the United States, Mexico, Europe and South America. PATIENTS: Over 9000 women and men aged 55 years and above at high risk for cardiovascular events such as myocardial infarction and stroke were recruite over 18 months. INTERVENTIONS: A 2X2 factorial design with ramipril and vitamin E with follow-up for up to four years. CONCLUSIONS: HOPE will be one of the largest trials of two new interventions to prevent myocardial infarction, stroke or cardiovascular death in high risk patients. The results of HOPE will have direct public health impact and are likely to be readily incorporated into clinical practice. Key design features of HOPE are inclusion of individuals at high risk of cardiovascular disease, inclusion of a substantial proportion of patients with diabetes (36%) and women (27%), and detailed substudies to provide data on mechanisms of benefit.

Publication Types:
- Clinical Trial
- Multicenter Study
- Randomized Controlled Trial

PMID: 8605634 [PubMed - indexed for MEDLINE]

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1: J Diabetes Complications 1995 Oct-Dec;9(4):318-22 Related Articles, Link

ELSEVIER SCIENCE
FULL-TEXT ARTICLE

Diabetic vascular hypertrophy and albuminuria: effect of angiotensin converting enzyme inhibition.

Allen TJ, Hulthen UL, Rumble JR, Jasik M, Cooper ME.

Department of Medicine, University of Melbourne, Austin, Australia.

The role of angiotensin-converting enzyme (ACE) inhibition with ramipril oı mesenteric vascular hypertrophy and urinary albumin excretion was explorec in a normotensive model of experimental diabetes. Serial measurements of albuminuria were performed in Sprague-Dawley control, diabetic rats, and diabetic rats treated with ramipril. Over 24 weeks, urinary albumin excretion showed a continuous rise in the untreated diabetic rats. Ramipril prevented the increase in albuminuria over the whole study period. After 6 months, animals were perfused with glutaraldehyde and sacrificed for measurement of mesenteric vessel wall/lumen ratio and kidney weight. Diabetes was associated with increased mesenteric wall/lumen ratio and kidney weight. ACE inhibition, despite no effect on glycemic control, attenuated mesenteric vascular hypertrophy but did not decrease kidney weight. In addition to the well-described renoprotective effects of ACE inhibition in diabetes, this class of agents may have a favorable effect on diabetic vascular disease.

PMID: 8573756 [PubMed - indexed for MEDLINE]

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Application#	Patent#	Status	Date Filed	Title	Inventor Name
06534392	Not Issued	161	09/22/1983	AMINOACID DERIVATIVES AND A PROCESS FOR THEIR PREPARATION	SCHOLKENS , BERNWARD
07222607	Not Issued	166	07/21/1988	BENZOTHIAZINONE DERIVATIVES, PROCESSES FOR THEIR PREPARATION PHARMACEUTICALS CONTAINING THEM, AND THEIR USE	SCHOLKENS , BERNWARD
07806634	Not Issued	166	12/13/1991	AZOLE DERIVATIVES, PROCESS FOR THEIR PREPARATION, AND THEIR USE	SCHOLKENS , BERNWARD
07512219	Not Issued	161	04/20/1990	RENIN-INHIBITING DIPEPTIDES, A PROCESS FOR THEIR PREPARATION, AGENTS CONTAINING THEM AND THEIR USE	SCHOLKENS , BERNWARD
07830355	5292755	150	01/31/1992	USPA BENZOLYGUANIDINES	SCHOLKENS , BERNWARD
09194749	Not Issued	161	12/03/1998	USE OF INHIBITORS OF THE CELLULAR NA+/H+ EXCHANGER (NHE) FOR PREPARING A MEDICAMENT FOR NORMALIZING SERUM LIPIDS	SCHOLKENS , BERNWARD
06477081	Not Issued	166	03/21/1983	CIS, ENDO-2-AZABICYCLO-(3.3.0)-OCTANE-3-CARBOXYLIC ACIDS, A PROCESS FOR THEIR PREPARATION, AGENTS CONTAINING THESE COMPOUNDS AND THEIR USE	SCHOLKENS , BERNWARD
06565900	5158959	150	12/27/1983	DECAHYDROISOQUINOLINE-CARBOXYLIC ACIDS	SCHOLKENS , BERNWARD
06658902	4727160	150	10/09/1984	METHOD FOR MAKING -2-AZABICYCLO-(3.3.0)-OCTANE-3-	SCHOLKENS , BERNWARD

				CARBOXYLIC ACIDS	
07392604	Not Issued	166	08/11/1989	6-AROYL-SUBSTITUTED 3, 4-DIHYDRO-2H-BENZOPYRANS, PROCESSES FOR THEIR PREPARATION, THEIR USE AND PHARMACEUTICAL PREPARATIONS BASED ON THESE COMPOUNDS	SCHOLKENS , BERNWARD
07151584	4999371	150	02/02/1988	SUBSTITUTED 3,4-DIHYDRO-2H-BENZOPYRANS, PROCESSES FOR THEIR PREPARATION, THEIR USE AND PHARMACEUTICAL PRODUCTS BASED ON THESE COMPOUNDS	SCHOLKENS , BERNWARD
07151488	Not Issued	166	02/02/1988	ALKYL-SUBSTITUTED N-BENZOPYRANYLLACTAMS, A PROCESS FOR THEIR PREPARATION, THEIR USE, AND PHARMACEUTICAL PREPARATIONS BASED ON THESE COMPOUNDS	SCHOLKENS , BERNWARD
07565270	Not Issued	166	08/10/1990	PEPTIDES HAVING BRADYKININ ANTAGONIST ACTION	SCHOLKENS , BERNWARD
07564618	5231083	150	08/09/1990	METHOD FOR THE TREATMENT OF CARDIAC AND OF VASCULAR HYPERTROPHY AND HYPERPLASIA	SCHOLKENS , BERNWARD
07266960	5169841	150	11/03/1988	RENIN INHIBITORS	SCHOLKENS , BERNWARD
07251168	Not Issued	166	09/28/1988	RENIN-INHIBITING DIPEPTIDES, A PROCESS FOR THE PREPARATION THEREOF, AGENTS CONTAINING THEM, AND THEIR USE	SCHOLKENS , BERNWARD
06565904	Not Issued	166	12/27/1983	AMINOACID DERIVATIVES, A PROCESS FOR THEIR PREPARATION, AGENTS CONTAINING THESE COMPOUNDS, AND THE USE THEREOF	SCHOLKENS , BERNWARD
06565887	5162362	150	12/27/1983	OCTAHYDROINDOLE-2-CARBOXYLIC ACIDS	SCHOLKENS , BERNWARD
08001221	5360791	150	01/06/1993	RENIN-INHIBITING AMINODIOL DERIVATIVES	SCHOLKENS , BERNWARD
07899122	Not Issued	166	06/18/1992	RENIN-INHIBITING AMINODIOL DERIVATIVES	SCHOLKENS , BERNWARD

07825829	5215968	150	01/28/1992	DIPEPTIDE DERIVATIVES HAVING AN ENZYME INHIBITORY ACTION	SCHOLKENS , BERNWARD
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07791502	Not Issued	161	11/14/1991	SUBSTITUTED AZOLES, PROCESS FOR THEIR PREPARATION, AGENTS CONTAINING THEM AND THEIR USE	SCHOLKENS , BERNWARD
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08445543	5684016	150	05/22/1995	METHOD OF TREATING CARDIAC INSUFFICIENCY	SCHOLKENS , BERNWARD
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07631418	Not Issued	166	12/21/1990	SUBSTITUTED 3,4-DIHYDRO-2H-BENZOPYRANS PROCESS FOR THEIR PREPARATION THEIR USE AND PHARMACEUTICAL PRODUCTS BASED ON THESE COMPOUNDS	SCHOLKENS , BERNWARD
07330042	5043344	150	03/29/1989	UNSATURATED N-BENZOPYRANYLLACTAMS	SCHOLKENS , BERNWARD
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